Date

Title : HALOTHANE EFFECT ON CAROTID SINUS BARORECEPTOR DISCHARGE IN HYPERTENSIVE DOGS

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Introduction: Studies in man have shown that halothane anesthesia depresses the baroreceptor reflex response. Other studies have shown that halothane increased the carotid sinus baroreceptor discharge but had no detectable effect on the aortic baroreceptor discharge rate. Few studies have been done to elucidate the mechanism of vascular depressant action of halothane in hypertension. The purpose of this study was to determine the effect of different concentrations of halothane on the carotid sinus pressure and the carotid sinus baroreceptor nerve action potentials in renal hypertensive dogs.

Methods: Conditioned female mongrel dogs were made hypertensive by placing a Goldblatt clamp on the left renal artery. The renal blood flow was reduced by 50% . Ten weeks were allowed for hypertension to be established. The dogs were then anesthetized with pentobarbital (25 mg/kg) and ventilated with 100% oxygen to maintain arterial PCO_2 at about 38-42 torr. The carotid sinus was isolated and the baroreceptor nerve was identified. The carotid sinus nerve action potential and lingual artery pressure were recorded simultaneously, before and 15 min. after different end-tidal concentrations of halothane in steps of 0,0.75, 1.5, 2.0, and 2.5% in oxygen.

Results: Figure 1 presents a typical record of the carotid sinus baroreceptor nerve action potentials (right-hand panel), heart rate and lingual artery pressure in response to different end-tidal concentrations of halothane. Under control conditions (0% halothane and 100% oxygen), the carotid sinus baroreceptor nerve discharge shows two distinct firing patterns: a larger amplitude one occurring during the systolic rise of the pressure pulse and a smaller amplitude one occurring during the dichrotic notch and the early diastolic period. As the concentration of halothane is progressively increased, there is a gradual diminution of the smaller action potentials corresponding to the diastolic period.

Discussion: It has been suggested that the baroreceptor resetting in hypertension may result from selective resetting of a larger fraction of the low stimulation threshold myelinated A-fibers and a smaller portion of the high threshold non-myelinated C-fibers. Our observations suggest that halothane at high end-tidal concentrations (1.5% and higher) reduces the nerve action

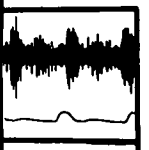
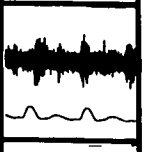
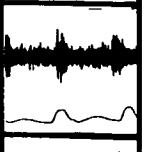
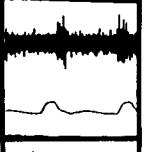
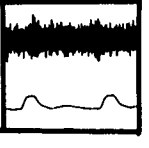
End-Tidal Halothane	Lingual Artery Pressure (torr)	Heart Rate (beats/min.)	
0%	210/165	150	
0.75%	190/155	185	
1.5 %	165/125	150	
2.0 %	152/115	133	
2.5 %	155/110	133	

Figure 1

potentials from the baroreceptor nerve C-fibers. This conclusion is based on the fact that halothane causes vasodilation, myocardial depression and hypotension, thereby lowering the pressure necessary for stimulation of both low- and high-threshold baroreceptors. However, because a greater number of A-fibers are affected in hypertension, lowering the blood pressure will silence more of the high-threshold receptors than the low-threshold ones. Thus, halothane effect may involve a selective reduction in stimulation of the high-threshold baroreceptors, with variable effect on the baroreceptor reflex, depending on the concentrations used. We feel that the effects of halothane reported here suggest that it may serve as a powerful tool for studying baroreceptor function under normal condtions and its adaptive changes in renal hypertension.

Supported in part by NIH Grant 1-ROl-HL 22667-02.

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